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November 4, 2011

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RE: Charles Krik vs. Weil-McLain
Eastern District Court of Pennsylvania

Dear Mr. Turner:

You have asked me to express an opinion regarding the role, if any, of Weil-McLain in causation of the medical condition in the above referenced litigation.

The following information has been provided by your office for review.

1. Plaintiff's First Response to Standard Interrogatories

2. Plaintiff's Verified Response to Standard Interrogatories

3. Plaintiff's Revised Statement of Expert Witness Reports

4. Deposition Transcript of Charles Krik dated 7/18/11

5. Industrial hygiene opinion report of Frank Parker CIH, CSP, PE, DEE

6. Report of D. Edwards P and D. Duvall PhD, PE. Engineering Systems Inc. Bakelite Exposures in CVLO Federal MDL Asbestos Cases. Report dated 10/18/2011

7. Report of Joseph Ferriter, dated October 18, 2011 [pipefitter]

8. Report of William LaPointe, dated October 14, 2011 [carpenter/millwright]

9. Physician medical records and reports including those of Drs.: A. Agha, Burhani, D. Cziperle, A. Frank, D. Frankel, J. Gallai, M. Jester, S. Klepac, A. Musabji, B. Patel, C. Reyes, A. Schonfeld, P. Tomas, D. Vanderkin

10. Hospital and clinic records including those of: Morris Hospital, St Joseph Hospital, Joliet Oncology Hematology Associates

11. Opinion reports of: A. Brody, B. Castleman

In March 2008, at age 71, Charles Krik presented with a spiculated left lower lobe nodule revealed on a CT scan done during surgical work for penile carcinoma. Left thoracotomy with left lower lobectomy provided the diagnosis of malignant adenocarcinoma consistent with bronchial alveolar tumor. There were focal areas of emphysematous bleb formation, and pleural tissue demonstrated pleural plaque. (Tomas report) His current condition had not been provided for review as of this date, over three years following diagnosis.

Radiologic evaluations demonstrated the left lower lobe nodule, left pleural effusion, and a left pleural thickening. There was no evidence of metastatic disease in the chest following the lobectomy. (Frankel, Patel reports) There were radiologic findings of emphysema and chronic obstructive pulmonary disease

(COPD). B reads reported bilateral pleural plaque and no parenchymal abnormalities. (Schonfeld reports)

Lobectomy was performed. However, pathologic evaluation of lung tissue for asbestos bodies or uncoated fiber burden was not reported.

Past medical history includes penectomy for invasive moderately differentiated squamous carcinoma, predominantly in situ, bilateral endoscopic sinus surgery, hypertension, lumbar scoliosis, diffuse changes of degenerative disc disease, left rotator cuff repair, bilateral hip replacement, and arthritis.

Charles Krik smoked cigarettes. It is reported in Answers to Interrogatories that he smoked one pack per day for 28 (28 packs/day•years), with cessation in 1982. (ANS) Medical reports indicated that Mr. Krik smoked 1 ½ packs of cigarettes a day for thirty years, to a level of 45 packs/day•years with cessation in 1982. (Burhani report)

Charles Krik reports a 46 year work life from 1954 to 2000 as a boilerman, boilermaker, pipefitter and inspector. Mr. Krik provided testimony regarding his occupational activities and associations with asbestos.

Date(s)	Approx Duration	Trade, Location	Description of testimony
1954 to 1970	16 years	Boilerman/ Boilermaker US Navy	Mr. Krik served as a boilermaker in the US Navy. He spent time aboard various ships including USS Sproston DD 577, Jenkins DD 447, Taylor DD 468, and Walker DD 517 (Fletcher class destroyers); USS Bryce Canyon AD 36 (Shenadoah class destroyer tender); USS Vulcan AR 5 (Vulcan class repair ship) and USS Tutuila ARG 4 (Luzon class internal combustion engine repair ship). (dep pg 14-16)
			The USS Bryce Canyon and USS Vulcan did repairs on destroyers. (dep pg 20) Mr. Krik worked on boilers and associated equipment in the fire and engine rooms. (dep pg 29) He testified that he removed insulation on equipment. (dep pg 31) The boiler shop replaced the insulation. (dep pg 33) Mr. Krik identifies Kaylo block insulation, CertainTeed pipe insulation, and Eagle 66. (dep pg 47) He pulled pumps apart and disturbed asbestos. (dep pg 52)
			Mr. Krik used sheet gasket material. He cut the sheet material with a ball peen hammer. (dep pg 55) The outside of the tanks were covered with block insulation with a finish cement. (dep pg 61) According to Mr. Krik when he opened the hatch he would tear up insulation. (dep pg 61) He would replace the insulation. (dep pg 62)
			On the USS Tutuila Mr. Krik was in charge of the valve shop. (dep pg 74) He tore down, repaired and tested valves. (dep pg 74)
1974 to 2000	26 years	Pipefitter Union Local 597, Hammond, IN	Mr. Krik was a union pipefitter and worked in various locations including refineries, chemical plants, schools, residences, manufacturing facilities, churches, car companies, steel plants, power plants and nuclear plants.

Date(s)	Approx Duration	Trade, Location	Description of testimony
			Mr. Krik testified that he cleaned up insulation debris with a dustpan and broom and put it in plastic bags. (dep pg 79, 131) He replaced damaged sections of pipe insulation. He would cut or knock the insulation off the piping." (dep pg 82) Mr. Krik worked on various brands of boilers. (dep pg 82-83) He would remove old boilers. He testified that he removed insulation around the boiler. (dep pg 96) He worked around other removing insulation during plant decommissioning. He was 25 to 30 feet away replacing gaskets during the insulation removal. (dep pg 129)
1976 to 1977	1 year	Boiler/Machinery Inspector Factory Mutual Insurance Company, Chicago, IL	During inspections of the boilers, Mr. Krik would have to crawl in and out of the boilers. (dep pg 84) The handhold, manhole gaskets and safety valve gaskets were made out of asbestos. (dep pg 85)

As to Weil-McLain boilers, Mr. Krik identifies Wei-McLain as a brand of boiler that he encountered. He specifically recalled installing one Weil-McLain boiler in a home. The boiler had 21 sections. (dep pg 217) They removed the old boiler and put a new Weil-McLain 21 section boiler in. Mr. Krik did not know the manufacturer of the old boiler that was removed. (dep pg 218-219) He describes the new boiler as 4 feet high by 3 feet wide and 6 inches thick from side to side. (dep pg 219) Mr. Krik states there was a trench around each section and he used black cement that Weil-McLain provided. He used asbestos rope or wicking between the sections. (dep pg 219) The black cement was a wet and premixed. (dep pg 222) The rope gasket came on a spool and he measured what he needed and cut it with tin snips. (dep pg 222-223) He used a small flat asbestos gasket on the hood. (dep pg 224) After 1980, Mr. Krik testified that he installed one other Weil-McLain boiler at a convent and repaired two Weil-McLain boilers at a manufacturing plant. (dep pg 226) The repair work consisted of welding cracks and replacing the sections. (dep pg 228) Mr. Krik handled gaskets during the repair. (dep pg 231)

It is now claimed that Charles Krik developed lung cancer, that asbestos exposure caused the malignancy, and that Weil-McLain boilers contributed to that exposure.

That Mr. Krik developed lung cancer is considered correct. The diagnosis was based upon appropriate radiologic and pathologic findings.

Cigarette smoking is a scientifically established etiology of lung cancer.

1. The causative bond between smoking and lung cancer has been established. (ACS, Alberg, Brownson, Doll, Halpern, Hecht, Peto 2000, UK/RCP, USDHEW, USDHHS) Mr. Krik smoked cigarettes to up to an estimated level of 45 packs/day•years.

2. Carcinoma of the lung is the primary cause of cancer mortality in the United States and is the single largest preventable cause of lung cancer in the world. It has been estimated that between 85% and 95% of deaths from lung cancer are directly attributable to smoking. (Hammond, Rodenstein, Rom, WHO) Fifty-five carcinogens have been identified in mainstream smoke

including twenty recognized pulmonary carcinogens. (Hecht)

3. Significantly, Mr. Krik had radiologic and pathologic findings of emphysema and chronic obstructive pulmonary disease (COPD). Cigarette smoking is the recognized etiology of these conditions. (Ingram, Pratt) This indicates cigarette habitation of causal significance for not only progressive benign lung disease, but lung cancer risk as well.

4. Depending on a number of factors, cessation of smoking typically reduces risk of malignancy, but in long term smokers risk persists. (Halpern, Hrubec, Peto, Reid, USDHHS) Cessation in this case (reported as 1982) did not eliminate the lung cancer risk

Cigarette smoking was the cause of the lung cancer in this case.

That occupational asbestos exposure contributed to lung cancer risk in this case has not been established. Guidelines, known as The Helsinki Criteria, have been applied to determine if asbestos has caused or contributed materially to lung cancer risk in this case. The probability of contribution increases as the exposure increases; cumulative asbestos exposure is considered the criterion for attribution of risk. Factors in determination of cumulative exposure include: asbestosis demonstrated clinically or histologically, an asbestos tissue burden within the range of values found with asbestosis, an estimation of 25 fiber/cc•years (f/cc•yrs) of asbestos exposure, or an occupational history defining heavy or moderate exposure over appropriate durations, and a latency period of ten years or more. (Helsinki 1997)

The presence of asbestosis (diagnosed clinically, radiologically or histologically) has significance as a surrogate for cumulative exposures and fiber deposition. (Henderson/Helsinki 2004)

1. Radiologic evaluations.

   a. Parenchymal changes. Mr. Krik lacks consistent structural changes (irregular opacities) by parenchymal imaging methods of asbestos related interstitial fibrosis. (ATS 1986, 2004)

   b. Pleural plaques. Radiologic evaluations demonstrated pleural plaques, sensitive hallmarks of past asbestos exposure. (ACR, Battifora, Craighead 1995, Hammar, McCaughey, Jones, Nishimura, Oury) Asbestosis and lung cancer risk are considered outcomes separate from pleural plaques. (ATS 2004) Plaques alone are insufficient to relate lung cancer to prior asbestos exposure. (Helsinki 1997)

   Mr. Krik lacks consistent radiologic evidence of cumulative asbestos exposure sufficient to materially contribute to lung cancer risk.

2. Histologic findings. Histologic criteria permit definitive diagnosis of asbestosis without requirement of clinical and radiographic data or exposure history. Such criteria include:

   a. "The histologic diagnosis of asbestosis rests upon the demonstration of peribronchiolar fibrosis and asbestos bodies in tissue sections" (Sporn and Roggli)

   b. "The demonstration of discrete foci of fibrosis in the wall of respiratory bronchioles associated with accumulations of asbestos bodies." (CAP/NIOSH)

Although lobectomy was performed evaluation of lung tissue for asbestos related changes and presence of asbestos bodies was not reported.

Asbestos contribution to lung cancer risk in this case was not established by histology.

3. Digestion studies: Analysis of lung digests can provide asbestos deposition and fiber type data to determine magnitude, and possibly sources of exposure. Such evaluation of asbestos bodies and uncoated fibers can establish association. (Helsinki 1997, Henderson/Helsinki 2004)

   Although lobectomy was performed and parenchymal tissue available, digestion study for asbestos bodies and uncoated fibers was not reported. Asbestos contribution to lung cancer was not established by digestion studies.

4. Occupational history. Testimony describes association with asbestos containing materials when Mr. Krik worked as a boilermaker and pipefitter. However, occupational history is neither substantive nor compelling as to direct, heavy, repeated asbestos exposure appropriate to development of lung cancer risk. The concept that any exposure to asbestos leads to increased risk of lung cancer is not supported by epidemiologic evidence. (Helsinki 1997, Henderson/Helsinki 2004)

Occupational association with asbestos products clearly occurred in this case, but has not been demonstrated as sufficient to provide contribution to lung cancer risk. Mr. Krik's lack of diagnostic radiologic findings, absence of appropriate histologic and fiber burden evaluation, and occupational history do not establish asbestos exposure as a significant contributory factor to lung cancer risk. The Helsinki Criteria for attribution of causation or material contribution to lung cancer risk by asbestos exposure are not met. (Henderson/Helsinki 2004)

Considering the above, the lung cancer in this case is not asbestos related.

However, even if the lung cancer in this case is erroneously deemed to be asbestos related, the assertion that Weil-McLain boilers created, or contributed to, lung cancer risk is not correct considering the inherent lack of contribution to dose and risk by sealing components, that exterior asbestos containing thermal insulation was not specified or provided, and lack of association of causal or contributory significance.

1. Inherent lack of contribution to dose and risk by boiler sealing components: It is unlikely that there could have been any contribution to risk by Weil-McLain boilers when dose generation potential is considered. Major factors to consider in dose generation are amount and accessibility of asbestos components, potential fiber release levels (exposure concentration) and time spent on maintenance (exposure duration). Dose is the product of intensity (concentration) and time (duration) expressed as fibers per cubic centimeter times years (f/cc•yrs) or fiber years. Dose determines risk.

   a. Meager use of asbestos materials. It is my understanding that asbestos materials in Weil-McLain boilers encountered during installation, inspection, and maintenance were confined to rope packing between sections, at access ports or doors and, a gasket at the burner flange.

b. Intensity (concentration): Competent air sampling studies of work with Weil-McLain boilers (Boelter & Yates reports), and gasket and packing studies (Boelter 2002, 2003, 2011; Liukonen; Mangold 2006), demonstrate that airborne asbestos concentrations, if even detectable, approximate typical background levels; well below both past and present OSHA permissible exposure limits. Thus, intensity of exposure of any potential significance to airborne fibers from such sealing component material is lacking.

c. Time (duration): Typically, any work with Weil-McLain boilers would be relatively brief, intermittent and infrequent, further reducing the dose factor of duration and possible dose accumulation of any significance.

The integration of meager use of asbestos materials, insignificant fiber release, and brevity of infrequent association precludes generation of dose with any asbestos related disease risk under typical association with internal sealing components in Weil-McLain boilers.

2. Exterior asbestos containing thermal insulation was not specified or provided. Although testimony places insulation on the Weil-McLain boiler removed from Mr. Kriks' home, it is my understanding that Weil-McLain did not specify or provide asbestos containing thermal insulation for their boilers. The risk of potentially significant fiber release associated with external thermal insulation could not have been introduced by Weil-McLain.

3. Lack of association of causal or contributory significance. Mr. Krik testified that he worked on four boilers he identified as Weil-McLain. He installed possibly two Weil-McLain boilers, and repaired two during his twenty six years as a pipefitter. Considering the factors above, his association with the boilers as described in testimony, and the limited number of Weil-McLain boilers he encountered, Mr. Krik's association with Weil-McLain boiler cannot be considered of causal or contributory significance for asbestos related disease risk.

These factors eliminate Weil-McLain boilers from rational consideration as contributing to risk of an asbestos related malignancy.

In summary it is my opinion to a reasonable degree of medical and scientific certainty that:

1. Mr. Krik developed lung cancer.

2. Cigarette smoking was the cause of the lung cancer in this case, and completely explains the lung cancer risk.

3. The Helsinki Criteria to determine if asbestos has caused or contributed materially to lung cancer risk are not met. The lung cancer in this case was not asbestos related.

4. Even if the lung cancer in this case is erroneously deemed to be asbestos related, Weil-McLain did not contribute to that risk considering:

a. inherent lack of dose and risk,

b. external asbestos containing thermal insulation was not specified or provided, and

c. lack of association of causal or contributory significance.

These factors remove association with Weil-McLain boilers from rational consideration as contributory to risk in this case.

5. Weil-McLain had no role in causation of, or contribution to, lung cancer in this case.

Between now and trial, I understand that I may be given an opportunity to review additional records, test results or expert reports relating to this case. Should any of this additional material influence my opinions, a supplemental report will be provided.

Sincerely,

Robert N. Sawyer, M.D.

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